TCT-353

Pathologic Intimal Thickening Plaque Phenotype - Not As Innocent As We Though. Study With 3D Intravascular Ultrasound And Virtual Histology

Tomas Kovarnik[1], Zhi Chen[2], Andreas Wahle[3], Hana Skalicka[4], Ales Kral[4], Stepan Jerabek[1], Milan Sonka[2], Jan Horak[5], Ales Linhart[6]
[1]General Teaching Hospital, 1st Medical Faculty, Charles University in Prague, Prague, Czech Republic, Prague, Czech Republic, [2]Dept. of Electrical and Computer Engineering, The University of Iowa, Iowa, IA, [3]The University of Iowa, Iowa, IA, [4]General Teaching Hospital, 1st Medical Faculty, Charles University in Prague, Prague, Czech Republic, Prague, Prague, [5]General Teaching Hospital, 1st Medical Faculty, Charles University in Prague, Prague, Czech Republic, Prague 2, Prague, [6]General Teaching Hospital, 1st Medical Faculty, Charles University in Prague, Prague, Czech Republic

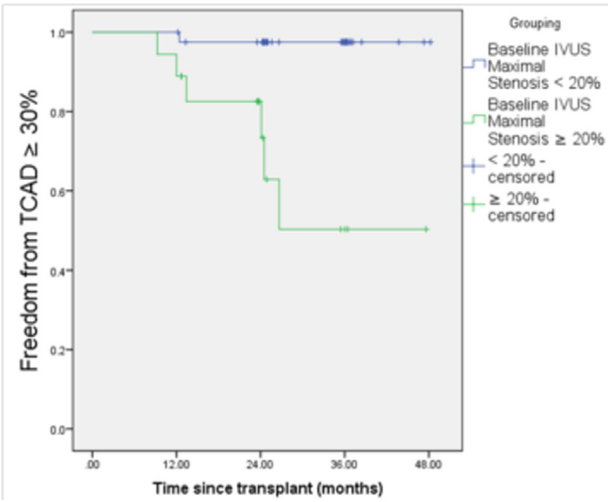
Background: Pathologic intimal thickening (PIT) plaque phenotype is an early stage of coronary atherosclerosis. It has been identified as a protective plaque phenotype in the PROSPECT trial. Unlike other trials that have used a frame-based definition for a plaque phenotype, we analyzed the vessel in 5 mm 3D segments of contiguous ECG-gated frames.
Methods: 32 patients with stable coronary artery disease and IVUS/VH data suitable for geometrically-correct 3 D reconstruction and quantitative analysis were retrospectively identified from a database containing 50 patients with baseline and 12 months follow-up IVUS of a non-culprit vessel. Volumetric and plaque-type indices were obtained from 5 mm long, adjacent, non-overlapping segments covering the entire length of the IVUS pullbacks. We analyzed total number of 280 segments of 5 mm length. The inclusion cut-off point for plaque was plaque burden >40%. Plaques were divided into four categories according to the AHA classification: pathologic intimal thickening (PIT), fibrous plaque (FP), fibro-calcified plaque (FcP), thick-cap fibro-atheroma (ThCFA) and thin-cap fibro-atheroma (TCFA). Risk score (RS) for all plaques was determined as follows: no lesion (NL) 0; PIT 1; FP 2; FcP 3; ThCFA 4; TCFA 5 points. Corresponding 5 mm vessel segments were compared between BL and follow-up.
Results: The mean change in RS for plaque phenotypes was: 0.48 for NL; 2.55 for PIT; 1.88 for FP; 1.2 for FcP; -0.06 for ThCFA; and, -0.57 for TCFA. The differences between changes of RS in PIT plaque phenotype versus other plaque phenotypes were statistically highly significant, except for comparison between PIT and FcP, where the p value was 0.08 (note that only 5 FcP segments were identified at baseline overall). New TCFA's and fibro-atheromas originated from PIT at rates of 53 % and 58.7%, respectively.
Conclusions: PIT is the plaque phenotype that exhibited the most significant changes of plaque morphology between baseline and follow-up and is a primary source of new fibro-atheromas.

TCT-354

Early Stenosis on Intravascular Ultrasound Immediately After Cardiac Transplantation Predicts Downstream Development of Transplant Coronary Artery Disease

Richard Cheng[1], Babak Azarbal[1], Aaron Yung[1], Jignesh Patel[1], Jon Kobashigawa[1]
[1]Cedars-Sinai Heart Institute, Los Angeles, CA

Background: Baseline to 1-year plaque progression on intravascular ultrasound (IVUS) predicts development of transplant coronary artery disease (TCAD). It is not known whether plaque on baseline IVUS immediately after cardiac transplantation predicts TCAD.
Methods: Between January 2010 and October 2011, routine left anterior descending (LAD) IVUS were performed on 59 transplant recipients. Maximal stenosis by area in percentage was measured in a blinded manner with quantification software. A ROC curve was performed to determine an optimal cutoff for freedom from angiographic significant TCAD ≥30%, and outcomes compared between the two groups.
Results: Mean age at cardiac transplantation was 53.6 ± 13.2 years. Baseline IVUS were done 45.1 ± 14.7 days after transplantation with an average segment length of 35.7 ± 5.0 mm. Average maximal stenosis by area was 18.5 ± 13.6%. Angiographic significant TCAD ≥30% was found in 7 of 59 (11.9%) patients; only 1 of 7 cases of TCAD was present at one-year angiography. Mean angiographic follow up was 30.7 ± 8.6 months. The area under the curve on ROC analysis was 81.6%, and an optimal maximal stenosis cutoff of 20% was chosen corresponding with a sensitivity of 85.7% and specificity of 76.9% to predict downstream TCAD. Maximal stenosis on baseline IVUS >20% predicted poor freedom from TCAD $p < 0.001$ (Log rank test). Interestingly, the majority of TCAD (57.1%) was predominantly non-LAD or diffuse disease despite IVUS only of the LAD.
Conclusions: Maximal stenosis seen on baseline IVUS ≥20% predicts downstream CAV even in non-LAD distributions.



TCT-355

Effect of Intravascular ultrasound-guided versus angio-guided percutaneous coronary intervention on clinical outcome: from the data of TRI retrospective registry

Young Jin Youn[1], Ji Hyun Lee[1], Jun-Won Lee[1], Junghan Yoon[1], Byung Ryul Cho[2], Kwang Soo Cha[3], Seung-Woon Rha[4], Kim Byung Ok[5], Min Su Hyon[6], Myung Ho Jeong[7], Sung Gyun Ahn[1]
[1]Yonsei University Wonju College of Medicine, Wonju, Korea, Republic of, [2]Kangwon National University College of Medicine, Chuncheon, Korea, Republic of, [3]Pusan National University Hospital, Busan, Korea, Republic of, [4]Korea University Guro Hospital, Seoul, Korea, Republic of, [5]Inje university, Sanggye-Paik hospital, SEOUL, Korea, Republic of, [6]Soonchunhyang University Hospital, Seoul, Korea, Republic of, [7]Chonnam National University, Kwang Ju, Korea, Republic of

Background: Intravascular ultrasound (IVUS) more accurately determines vessel size and lesion severity and estimate a detailed plaque composition than conventional coronary angiography. But there is controversy whether use of IVUS for treating coronary artery disease (CAD) with percutaneous coronary intervention (PCI) improves better clinical outcome.
Methods: Among 4,980 consecutive patients underwent PCI for CAD were analyzed from the TRI retrospective registry, data from 4,466 patients after excluding the patients with inhospital mortality and inappropriate data for PCI and clinical outcome were analyzed. The patients were divided into two groups whether IVUS was used (IVUS-guided PCI group; n = 1,191) or not (angio-guided PCI group; n = 3,275) during PCI. Major adverse cardiac event (MACE) was a composite of any death, myocardial infarction and any revascularization at 1-year of clinical follow-up.
Results: Age was similar between two groups (64.3 ± 11.5 years in angio-guided PCI group vs. 64.7 ± 10.5 years in IVUS-guided PCI group; $p = 0.297$) but male were dominant (67.3% vs. 63.1%; $p = 0.011$). History of hypertension (55.4% vs. 59.7%; $p = 0.012$), diabetes (30.4% vs. 33.8%; $p = 0.034$), smoking (39.8% vs. 51.6%; $p < 0.001$), and previous myocardial infarction (MI) (4.5% vs. 7.8%; $p < 0.001$) were frequent in IVUS-guided PCI group. The rate of stable angina (15.4% vs. 20.8%; $p < 0.001$) was higher but the rate of acute MI (39.5% vs. 31.2%; $p < 0.001$) was lower in IVUS-guided PCI group. Discharge medication was similar except use of clopidogrel (98.1% vs. 99.2%; $p = 0.011$), beta blocker (63.0% vs. 69.7%; $p < 0.001$), and statin (84.9% vs. 81.2%; $p = 0.004$). In IVUS-guided PCI group, more number of stent (1.6 ± 0.9 vs. 1.9 ± 1.2; $p < 0.001$) with larger diameter (3.1 ± 0.4 mm vs. 3.2 ± 0.4 mm; $p < 0.01$) and longer length (38.2 ± 24.0 mm vs. 45.2 ± 32.1 mm; $p < 0.001$) were used. The incidence of MACE (6.1% vs. 7.0%; $p = 0.298$) and stent thrombosis (0.7% vs. 0.9%, $p = 0.565$) were similar between two groups. On multivariate analysis, the use of IVUS was not a factor for MACE.
Conclusions: In our retrospective registry, IVUS-guided PCI in patients with CAD did not improve the clinical outcome and stent thrombosis.